9

Arguing about Group Selection

The Myxoma Case

1. GROUP SELECTION IN THE WILD

In the previous chapter, I mentioned the distinction between earlier forms of group selection, such as those associated with the Chicago School of ecology and V. C. Wynne-Edwards, and more recent forms of group selection stemming from David Sloan Wilson's work on trait groups. One of the contrasts between these two traditions lies in the degree of mathematical rigor within each. The mathematical modeling of group selection that currently exists itself has followed two relatively distinct traditions. The first is a laboratory or experimental tradition with its roots in Sewall Wright's work on evolution in structured populations and exemplified by Michael Wade's work over the past 25 years at Chicago and Indiana. The second is an adaptationist tradition that, while keeping "old" approaches to group selection at arm's length, has sought to use its mathematical sophistication and philosophical savvy to overturn the influential challenges to group selection issued by the rise of the gene's eye view of natural selection.[1]

One limitation that both traditions face is that they can be seen as "merely theoretical" by their opponents. Indeed, despite attention within these traditions to the practical or applied side of their theoretical work, such a perception is common amongst evolutionary biologists who are not specialists in the area. Group selection might be brought about through artificial means in a laboratory, or might receive an adequate, robust mathematical and philosophical justification, but what about the real world? What is the evidence that groups are the agents of selection in the wild?

A standard example often given of group selection in the wild is the spread of the myxoma virus after it was introduced in Australia in 1950 in order to control the rabbit population there. The virus causes myxomatosis, a disease that can prove extremely lethal, killing rabbits often within a matter of days. Richard Lewontin introduced the myxoma case as an example of group selection that was likely to be widely instanced, and this view of the evolution of myxoma has been developed further by Elliott Sober and David Sloan Wilson. Those skeptical of group selection have provided alternative interpretations of the case. Sober and Wilson have replied to some of these, yet despite my sympathies with many of the views that Sober and Wilson defend, I also think that what one should say about the myxoma case is less clear cut than Sober and Wilson seem to think it is.[2]

The example can also be used to raise some general issues about the nature of selection and how it is usually conceived. One such issue is the place of test cases in science, and the resolvability of scientific disputes, such as that over the agents of selection.

2. TEST CASES AND RESOLVABILITY

By virtue of their controversial or novel nature, some views and theories carry a special burden of proof. A dialectically powerful way to discharge that burden is to present a clear and decisive example that can only or can best be explained by the view or theory. In science, such examples take the form of phenomena that serve as test cases for the view or theory under consideration.

The theory of group selection is one such view. As I mentioned in the previous chapter, David Sloan Wilson and Elliott Sober have been at the forefront of the revival of group selection within evolutionary biology. Amongst the theoretical work that they have done in articulating group selection as a coherent and potentially powerful force directing evolution, and the empirical evidence they have adduced in support of the efficacy of group selection in actual populations, are several examples they represent as test cases of their theory. In their recent *Unto Others*, they present two such examples: the evolution of female-biased sex ratios, and the evolution of virulence.[3]

In this chapter, I shall focus exclusively on the latter case, particularly on the most detailed example they discuss, that of the evolution of avirulence in the myxoma virus. There are two immediate conclusions that I shall argue for from an examination of this case, and a third that

warrants further discussion. These conclusions carry with them a number of broader implications for how we do and might think about biological reality that I discuss further in Chapter 10. The immediate conclusions themselves are of increasing generality, moving from a weaker to a stronger claim about the myxoma case, and then in turn to the debate over the levels of selection more generally.

First, as described in the philosophical literature on the agents of selection, the myxoma case does not, contra Wilson and Sober, "provide compelling evidence for group selection." It is not the decisive test case of group selection theory that they claim it is. This is not simply because there are possible, competing individual-level explanations of the case, but, as I will argue in sections 3 and 4, because there is no independent basis for preferring the group selection account of the phenomenon to alternative explanations.[4]

Second, the myxoma case is likely to remain subject to multiple, alternative explanations, only some of which appeal to group selection. This second conclusion goes beyond the first not only in strength, but also by taking us beyond the abbreviated characterizations of the myxoma case that have featured in the debate over the agents of selection into the epidemiological and virological literatures in which myxomatosis is discussed more fully. Given standard views of how scientific disputes are rationally resolved, one could reasonably expect the addition of further empirical details to the case to reveal the true level or levels at which the evolution of virulence is governed by natural selection. Yet far from achieving this goal, restoring some of the complexities to the myxoma case usually omitted or glossed over by philosophers and biologists alike reinforces and strengthens the first conclusion. The myxoma case is likely to be irresolvable vis-à-vis the debate over the agents of selection. Defending this more general conclusion will be my focus in sections 5–7.

Third, what is true of the myxoma case is true more generally, and so the overall debate between proponents of group selection and other agents of selection is also likely to be irresolvable. Invoking the metaphor of viewpoints or standpoints, many evolutionary phenomena that can be adequately explained by invoking one viewpoint – whether it be that of the gene, the individual, or the group – can be explained adequately by invoking at least one other viewpoint. A defense of this final claim would involve at least a detailed examination of the other test case for group selection theory that Wilson and Sober provide, that of the evolution of female-biased sex ratios. And a version of this generalization that treated various levels of selection symmetrically would require exploring other

phenomena, such as meiotic drive, that have been claimed by many to constitute decisive cases vis-à-vis the theory of *genic* selection.[5]

A strong form of this third conclusion is entailed by a widespread form of pluralism about the levels of selection that maintains that these viewpoints are, in an important sense, equivalent. But my argument in this chapter does not rest on pluralism about the levels of selection. Indeed, in Chapter 10, I shall explore pluralism more fully and argue against several forms of pluralism that have recently gained some currency in the literature on the levels of selection.[6]

I begin in the next section with a familiar sketch of the myxoma case and the debate over it, going on in section 4 to argue against the most commonly cited reasons for resolving this debate one way or the other. Included here is the appeal that Sober and Wilson make to "the averaging fallacy" in criticizing individualistic explanations of the myxoma case. This fallacy has been invoked by these authors, separately and together, for many years in their defense of group selection. In keeping with the general themes of this chapter, I shall argue that attributions of the fallacy do little to support the idea that the myxoma case is a paradigm case of group selection in the wild.

3. THE CASE OF THE MYXOMA VIRUS: A SKETCH AND TWO INTERPRETATIONS

The myxoma virus was introduced into Australia as a way to control an exploding rabbit population in 1950, a solution that met with unexpected limits to its success. After myxomatosis initially killed 99% of the hosts infected, and seemingly quickly, the rabbit population began to rise steadily again. As expected from individual selection theory, rabbits from the wild tested against laboratory strains of the virus had an increased resistance to the virus. But it was also found that the virus in the wild had decreased its virulence, compared to those very laboratory strains, a finding anomalous within the theory of individual selection.

In Australia in 1950, the virus was primarily transmitted between host rabbits by an arthropod vector, the mosquito. As Lewontin notes, "mosquitoes do not bite dead rabbits." Thus, the transmission of the virus from dead hosts is minimal. More virulent forms of the virus kill the host organism more quickly, and thus are less likely to be transmitted to other host organisms. Less virulent forms of the virus tend to fixate on the overall host population of rabbits, with more virulent forms disappearing. The chief phenomenon to be explained is the apparent evolution of

what is often termed "avirulence" in the myxoma virus. Why did avirulent strains of the virus predominate? What was the mechanism that brought this observed result about?[7]

The standard group selectionist interpretation and explanation of this phenomenon is as follows. Within single host organisms, more virulent forms of the virus are favored, since these have a higher reproductive rate, and come to replace less virulent strains within any given rabbit. Thus, higher virulence is fitter within an organismic host than is lower virulence and evolves by individual selection. But forms of the virus with lower virulence are likely to infect more hosts by allowing their hosts to live longer, and so are fitter across organismic hosts. In effect, viruses on a given rabbit are a trait group in both senses discussed in the previous chapter: they are "populations enclosed in areas smaller than the boundary of the deme," and sets "of individuals that influence each other's fitness with respect to a certain trait but not the fitness of those outside the group." If we assume that an individual rabbit delineates a group of viruses, then this implies that low virulence is altruistic, and that it evolves because of the higher fitness of less virulent groups of viruses. This is a process of selection between groups, and thus an example of group selection.[8]

There is also an obvious, contrasting individualist explanation for the phenomenon, one provided by Douglas Futuyma and Richard Alexander and Gerald Borgia. A viral strain that kills its host is less fit than one that does not kill its host. Low virulence in a viral strain reduces the chance that it will kill its host. Thus, low virulence is an adaptation of individual viral units, and it evolves by a process of selection that acts on individuals. As a result, lower levels of virulence spread through the population of viruses as a whole via individual selection.[9]

On this view, the only relevant groups are the overall population of rabbits and the overall population of viruses. Individual selection changes the composition of the latter of these from one with predominantly highly virulent individuals to one with predominantly less virulent individuals over evolutionary time. In positing low virulence as an adaptation of individual viruses, this view also denies the existence of low virulence as an altruistic trait, since low virulence is depicted as being to the evolutionary advantage of the individual virus.

Sober and Wilson have made two related criticisms of this sort of individualistic interpretation, criticisms that have a longer history. First, it involves what they call the *averaging approach* to calculating fitness: it calculates the fitness of traits across the entire metapopulation, rather than within the constituent populations, that is, groups. Sober and Wilson regard the averaging approach to fitness in this context as involving a

fallacy, the *averaging fallacy*. As a fallacy, this is the mistake of using the averaging approach to fitness to draw a conclusion about the level of selection that governs its distribution. This inference is a fallacy, because in adopting the averaging approach, one collapses the distinction between within- and between-group selection, itself crucial to characterizing, respectively, the process of individual selection and that of group selection. Thus, once one adopts the averaging approach, no inferences can be validly drawn about the level at which selection operates. Second (and subsequently), by focusing exclusively on the products of natural selection – overall fitness values – this sort of interpretation ignores the processes that generate those products. The debate over the agents of selection, however, is precisely one over how natural selection operates. Thus, the individualistic explanation begs the question at issue.[10]

Individualistic explanations of the myxoma case have been motivated by a general concern over the coherence or prevalence of group selection, one that turns, as I indicated in the previous chapter, in part on doubts about the ontological status of groups. Recall the arbitrariness problem: that there is no way to demarcate groups of organisms from mere sets of organisms, thus implying that any n-tuple of individuals could be said to constitute a group. And the ephemerality problem: that groups, unlike individuals or genes, are not sufficiently permanent arrangements in the biological world to be the units on which natural selection operates over evolutionary time. Both problems reflect a concern over the ontological status of groups insofar as they express the view that groups are "not real" in the way that genes and individuals are. Such existence as they have makes them unsuitable to play the role of an agent of selection in general, and in the myxoma case in particular.

4. THE STATE OF THINGS: AVERAGING AND THE STATUS OF GROUPS

If the individual selectionist account of the myxoma case were to commit a fallacy, as Sober and Wilson claim, then clearly that would provide a reason to favor the group selectionist account of it. And, conversely, if the group selectionist account of the myxoma case were to suffer from either the arbitrariness or the ephemerality problem, then the apparent gridlock between the two explanations would be broken in favor of the individual selection account. It is important to recognize that neither resolution of the debate over the myxoma case is plausible.

Consider first the claim that the group selectionist account of the myxoma case suffers from the arbitrariness and ephemerality problems,

and that this provides a conclusive reason for preferring the individual selectionist account. Neither charge holds of the groups of viruses bounded by individual host organisms. A rabbit-bound population of viruses constitutes an entity that faces shared ecological and evolutionary circumstances. Individual viruses in such a group draw on the same resources, and by virtue of physical proximity to one another, face the possibility of host resistance or vector transmission together. Importantly, populations of viruses on different rabbits are likely to differ from one another in these respects, and so differ in fitness. Thus, rabbit-bound groups of viruses are not arbitrary from an evolutionary point of view (cf. section 6 that follows, however). Furthermore, given that typically there will be hundreds of generations of viruses on any given rabbit, these groups are not ephemeral either. In short, even if there *are* putative instances of group selection that are faced with the arbitrariness and ephemerality problems, group selection in the myxoma case cannot be rejected because the case invokes arbitrary or ephemeral groups.

Consider now the averaging fallacy. There are two responses that can be made to the charge that such a fallacy is committed in the individual-level explanation of the myxoma case. The first of these concedes that the inference from adopting the averaging approach to fitness to a conclusion about the level at which selection operates is a fallacy, but denies that this inference is made in explaining the myxoma case. The second response argues that the attribution of the averaging fallacy in this case, and perhaps more generally, itself begs the question against the proponent of individual selection. Let us take each in turn.

If the averaging fallacy is committed in the individual level explanation of the myxoma case, then that explanation must somewhere invoke the averaging approach to calculating fitness. Thus, whether the averaging fallacy is committed turns on what basis there is for the claims that:

1. A viral strain that kills its host is less fit than one that does not kill its host.
2. Low virulence reduces the chances of killing one's host.

Thus,

3. Low virulence is an adaptation of individual viruses.

So,

4. Low virulence evolves by individual selection.

For the averaging fallacy to be committed here, the fitness explicit in (1) and implicit in (3) must be conceptualized or calculated as an average over the whole population of viruses. Furthermore, this averaging must be the reason for holding at least one of (1)–(4) to be true.

There are, however, at least two related reasons for thinking that (1)–(4) are true that are independent of whether fitness is calculated over the whole population. Both reasons pack some dialectical punch in part as a result of the distance that proponents of the "new" group selection have self-consciously promoted between their views and the "old" group selection tradition featuring Emerson and Wynne-Edwards.

First, it has been well known, at least since the work of David Lack in the 1950s on optimal clutch size in birds, that individuals can increase their reproductive success by limiting the number of offspring they produce. One might think that reduced virulence in individual viruses is simply an instance of such a reproductive strategy, and so an adaptation of individual viruses that reflects the increased fitness of those viruses. Explanations such as Lack's have not been seriously disputed by proponents of group selection as appropriate in at least some cases. This suggests that it does not commit the averaging fallacy. Given that, there would have to be some relevant difference in the myxoma case that vitiates the same reasoning that Lack used, were any "averaging fallacy" charge to be plausible in that case.[11]

Second, since the fitness of any trait needs to be relativized to an environment, low virulence may be viewed as having higher fitness once we consider an environment in which host death constitutes a major limitation on long-term reproductive success. One comes to view lower virulence as an adaptation by considering the nature of this environment more fully, rather than simply by defining fitness and adaptation as "what evolves." Again, there is a basis for (1)–(4) that appeals to points that group selectionists themselves appear to be committed to.

Such reasons are independent of whether we calculate fitnesses within or across local groups of viruses (= rabbits) in that they are factors "in play," so to speak, prior to any calculations of fitness at all. Of course, if the individual selectionist viewpoint is adopted, then fitnesses *will* be calculated across the whole population of viruses. But provided that this is not the basis for adopting (1)–(4), the averaging fallacy has not been committed.

So much for the first response to the averaging fallacy charge. The second response is more dialectically powerful in the context of the overall argument of this chapter. The idea behind the averaging fallacy charge is

that individualists illegitimately collapse the distinction between within- and between-group fitnesses by averaging across groups to begin with. But one can collapse this distinction (illegitimately or not) only if the distinction is already there to collapse. This in turn presupposes not simply that there are groups of viruses (= rabbits) in the overall population, but that these groups constitute a causal structure to which natural selection itself is sensitive. Yet this is precisely what proponents of the individual level explanation will deny – not necessarily in general but in this particular case. For although there is a sense in which rabbits are groups of viruses, it is not this group structure on which natural selection operates. Rather, it operates on individual viruses, viruses whose fitness is sensitive to features of their environment, including the nature of their hosts (= groups of viruses) and limitations on transmission imposed by the nature of the vectors that mediate the spread of the virus. In short, an averaging fallacy can be committed only if we already concede that groups themselves are acted on by natural selection, and this is precisely what is at issue between proponents of the two explanations. Ironically, it is Sober and Wilson's attribution of an averaging fallacy itself that begs the question against the individualist.

To summarize: Sober and Wilson argue that at least some individualistic views of the myxoma case commit the averaging fallacy, and that interpretations that rely crucially on a misplaced skepticism about the reality of groups (of viruses, in this case) should be rejected. I concur. But in generalizing this claim and presenting the myxoma case as a test case for the theory of group selection, a test that the theory passes, Sober and Wilson are mistaken. The test can also be passed by the theory of individual selection. Moreover, their general invocation of the averaging fallacy itself begs the very question at issue. At this point, I draw my first conclusion: that the myxoma case, as it is usually presented, is not a decisive example of group selection.

I have already remarked that the dispute over the myxoma case has involved an abbreviated characterization of the case. One healthy suspicion fostered by interplay between the philosophy, history, and social studies of science over the last forty years is directed at just this sort of impoverished treatment of actual examples. Might our anemic characterization of "the evolution of avirulence" in myxoma actually create or maintain the standoff in the debate over it? Shouldn't a more complete rendering of the facts allow a resolution of the apparent deadlock between proponents of our two perspectives?

In the next section, I fill out the picture of the pathology of the disease and the nature of its transmission, drawing largely on the masterful book-length treatments of Fenner and Ratcliffe and Fenner and Fantini. I shall argue that not only does a more complete rendering of the case fail to challenge my conclusion about the irresolvability of the case, but that it actually reinforces that conclusion in a way that suggests the pair of stronger conclusions about irresolvability stated in section 2.[12]

5. A LITTLE MORE ON THE VIROLOGY AND EPIDEMIOLOGY OF MYXOMATOSIS

The myxoma virus is a member of the family *Poxviridae*. The disease it causes in rabbits was first described by the Italian medical researcher Guiseppe Sanarelli in 1898 when European rabbits he received for his laboratory work in Uruguay (sent from Brazil) developed the disease. The virus is relatively host specific, being largely restricted to a variety of "rabbit species," including those from the *Sylvilagus* and *Oryctolagus* genera, commonly called, respectively, the Californian and European rabbit. The strain of the virus introduced into Australia was isolated from an *Oryctolagus* rabbit in Brazil in 1911. This strain was maintained through serial passage from host to host until it was introduced to Australia in 1950.

At the initial site of myxoma infection in rabbits a skin lesion appears, and the virus begins replicating in the body of the host, working its way through lymph nodes and the circulatory system. Secondary lesion sites also appear, and there is often a discharge from the eyes and a swelling of the genitalia. The preferred concept of virulence used by epidemiologists is host-fatality or lethality, the percentage of infected rabbits that die, though this is commonly estimated from a more readily operationalized measure, the average number of days to death. An estimate was necessitated in Australia by the impracticality of keeping large numbers of rabbits, and was made possible by the apparent correlation and regression coefficient between case-mortality rate and the more readily measured-in-the-lab variable of average number of days to death.

With the original strain of the virus introduced into Australia, infected laboratory rabbits survived about five days, making the virus extremely lethal. In one of the first systematic field counts, at Lake Urana in 1951, the lethality of the virus was estimated at 99.8%, this being reduced to 90% in 1952.[13]

TABLE 9.1. *The virulence of myxoma strains sampled in Australia, 1950s*

Virulence grade	I	II	III	IV	V	
Degree of virulence	Extreme	Very high	Moderate	Low	Very low	
Mean survival time (days)	<13	13–16	17–28	29–50	–	
Case-fatality rate (%)	99.5	95–99	70–95	50–70	<50	Number of strains tested
1950–51	>99					
1951–52	33	50	17	0	0	6
1952–53	4	13	74	9	0	23
1953–54	16	25	50	9	0	12
1954–55	16	16	42	26	0	19
1955–56	0	3	55	25	17	155
1956–57	0	6	55	24	15	165
1957–58	3	7	54	22	14	112
1958–59	0.5	20	57	14	8	179

Source: Modified from Table 7.2 of Frank Fenner and Bernardino Fantini, *Biological Control of Vertebrate Pests: The History of Myxomatosis – An Experiment in Evolution* (Oxford: CABI Publishing, 1999).

Because the concept of virulence used here is relational, this reduction in virulence alone could in principle be accounted for by a change in host resistance in the population. But suspecting that the virus had changed, researchers checked virus serum taken from infected rabbits against captive rabbits that had been isolated from the virus until that point. They found, indeed, that the virus itself was reduced in its lethality. In their attempts to measure the changes in the virulence of the virus itself, from 1951 researchers began gathering multiple samples of the virus from wild populations of infected rabbits. By 1959 they had 672 such strains (no doubt, not all distinct), and they introduced five grades of virulence for these, rated by their estimated case-mortality rates, starting with >99% for Grade 1, the original strain (rarely recovered from the wild), through to <50% for Grade V. The virus stabilizes at Grade III, with this constituting roughly 50% of the strains throughout the 1950s, with an estimated lethality of 70–95%. As can be seen in Table 9.1, the incidence of Grade II viruses (lethality: 95–99%) remains over 10% of the sampled population for much of the period surveyed, and increases significantly from 1958 to 1959. The incidence of Grade V viruses remains extremely low throughout, and Fenner and Ratcliffe note that "it

is unlikely that a virus of this type would survive in nature." Simple talk of "the evolution of avirulence" neither conveys nor does justice to the complexities here.[14]

There is a further complication to the basic finding that less virulent strains have evolved. The system of viral grades, the categorization of particular strains under them, and the resulting distribution pattern that changes over the years, all presuppose the correlation between mean survival time and survival rate mentioned a few paragraphs ago. Work led by Ian Parer has recently challenged this presupposition, and called into question whether the change in virulence was as radical as suggested by data such as that represented in Table 9.1.

First, Parer and his coworkers found a sire effect of 20–25% in offspring immunity, where sires had been infected with myxomatosis less than ten months prior to the birth of their offspring. The myxoma virus is disproportionately accumulated in the testes of wild rabbits, with its DNA remaining there long after infectious myxoma virus is no longer found there (or in other tissues). As they say, "the sire effect is likely to have contributed to the observed initial rapid increase in the resistance of rabbits," and to account for some of the variation in measured virulence from year to year.[15]

Second, Parer points out that the use of mean survival times as a measure of virulence has systematically underestimated the lethality of the myxoma virus. Parer was prompted to investigate the relationship between mean survival time and survival rate (lethality) after finding that most field strains of the virus were of high lethality (Grade I). Basically, the initial regression coefficient between the two was artificially high, due to the inclusion both of one highly attenuated strain of virus and of estimated survival times for rabbits surviving beyond fifty days. Once these are excluded and the relationship between mean survival time and lethality recalculated, the regression slope for these two variables shifts from .67 to .23. This means that although the correlation between the two remains strong, small variations in mean survival time correlate with large variations in lethality, and so the small errors in the former can lead to large errors in the latter. Parer argues that relying on an inflated regression between mean survival time and lethality "has resulted in most field strains being allocated inappropriate grades and it has distorted to some extent our perceptions of the types of evolutionary changes that occurred after the myxoma virus was introduced into Australia." The full significance of these findings remains subject to further investigation.[16]

Transmission is also more complicated than the standard picture from section 3 suggests. While the most prevalent and far-reaching form of transmission is via an arthropod vector, the virus can also be transmitted through direct contact or contagion between hosts, or through nonanimate vectors, such as thornbushes or warrens. Given that rabbits live in close proximity to one another in hutches, these are actual (not merely possible) mechanisms for transmission. Arthropod vectors pick up the virus on their mouthparts when they feed on an infected host animal, particularly at a lesion site, and then transmit the virus when they move to another host, or to another part of the same host. Vectors preferentially feed on the head of the rabbit, perhaps because it is the primary site of lesions. Thus, as well as between-host transmission of the virus, there is within-host transmission facilitated by vectors themselves and by the host's circulatory system, and differential vector transmission within a host.

Transmissibility of the virus by arthropod vectors, such as the mosquito, depends on facts about the ecology and life cycle of those vectors. The concentration of mosquitoes varies in accord with heat and moisture, and thus their abundance follows a seasonal cycle. This represented a problem for the use of myxoma in controlling the wild population of rabbits in Australia, not only in areas in which the concentration of mosquitoes was insufficient to transmit the disease effectively at any time of the year (for example, in the drier plains of Western Australia), but in all areas during the winter months. In the late 1960s, in response to this problem, the Australian government explored possibilities for introducing another vector for transmission, the European rabbit flea (*Spilopsyllus cuniculi*) that infests rabbits and does so year round. *S. cuniculi* was, in general, an effective vector for transmitting the myxoma virus throughout the year, and was particularly effective in infecting young rabbits, which significantly reduced the population growth for the following year. However, *S. cuniculi* survived poorly in arid conditions, and in the early 1990s another flea species, *Xenopsylla cunicularis*, was introduced from Spain.

The introduction of vectors alternative to the naturally occurring mosquito was thus aimed primarily at solving what has been called the *problem of overwintering*. Due to vector reduction during the winter months, more virulent forms of the myxoma virus could not survive the winter: they killed their hosts before being spread to other hosts. Fenner and Ratcliffe identify the occurrence of attenuated strains in geographically distinct areas as a reason to think that the virus was mutating within local populations, rather than being vector-transmitted there, since it was unlikely that vectors could reach these areas from regions in which

attenuation had already taken place. The switch in vectors met, however, with mixed and limited success as a way to control the rabbit population, a point I shall return to in the next section.[17]

Finally, given that the attention commanded by the myxoma case amongst those interested in the mechanisms of *natural* selection derives in part from its "in the wild" status, it is relevant that the bulk of the long-term data reported is the result of the natural overwintering of the virus in rabbit populations together with supplemental "inoculation campaigns." This program of human-induced infection was systematic and sustained, involving direct transmission of the virus by human beings (researchers and farmers) to over 20,000 rabbits on average in each of the twelve years following its original introduction. In addition, farmers intervened in another way in engaging their fondness for shooting and poisoning rabbits, which had become a major threat to their livelihood. Thus, human agents play a key role in the spread and maintenance of myxomatosis, not just through the selection of additional vectors, but also by serving as initiating vectors for transmission of the virus and in culling the (infected) rabbit population. This is what one would expect in a case that is, after all, a form of pest control. Whether these facts impugn the status of the myxoma case as one of group selection *in the wild*, I leave as an exercise for the reader.[18]

6. WHY FURTHER DETAILS REINFORCE THE IRRESOLVABILITY CLAIM

There is a surprisingly large gap between the epidemiological literature on myxomatosis and the evolutionary debate that the phenomena it studies has inspired. The tendency in virology and epidemiology is to focus on the documented findings in natural populations of rabbits via sampling techniques, approaching the question of the mechanisms mediating changes here through laboratory studies of both rabbit and virus. There has been little explicit attention to the question of just how natural selection operates in this case. For example, Fenner and Ratcliffe attribute the reduction in virulence they document as "being due to mutations of the virus which were subsequently selected for," but do not specify the selective mechanism. Later, in discussing the evolution of attenuated strains of the virus, they appeal to both the greater survival over the winter months of rabbits with attenuated strains of the virus and to lower temperatures during the winter as facilitating this survival (of both rabbits and virus). Again, the nature of the selective mechanism

itself is not further specified. In the extensive overview that Fenner and Fantini provide of the history of myxomatosis, this question of the level at which selection operates remains not simply unanswered, but (to a close approximation) unasked.[19]

General textbook treatments of the example have sometimes taken the "classic view" of the evolution of lower levels of virulence to be group selectionist. I have found it difficult, however, to find such a view – indeed, any explicit view on the agents of selection issue – in Fenner and Ratcliffe and Fenner and Fantini. Those involved most directly in the field and laboratory work on the myxoma virus seem to have individual selection, if anything, in mind as the form that natural selection takes. This is largely because of the emphasis on a range of factors that affect the spread of lower levels of virulence – host resistance, temperature, means of transmission – all discussed in terms of their effects on individual fitness.[20]

Consider, by contrast, the literature on the levels of selection that appeals to the myxoma example. Typically describing the example in a short paragraph or so, the details sketched previously are omitted. In particular, transmission by contagion is ignored, as are secondary lesion sites and preferential vector feeding, the effect of which is to highlight the significance of vector-mediated rabbit-to-rabbit transmission, that is, transmission between groups of viruses. For group selectionists, population structure "pops out" as a salient cause of the spread of what is observed to evolve, that is, lower levels of virulence. By contrast, although individualists ascribe a role to population structure, as we will see later in this section, they oppose this interpretation on quite general grounds. They subsequently see little need to appeal more than minimally to the details of the case, being concerned primarily to debunk the claims of group selectionists.

A range of putative examples of decisive empirical findings, actual or possible, is suggested by the details sketched in the previous section. These include those from studies that artificially increase (or decrease) the respective strengths of individual and group selection in an experimental group, and the examination of evolutionary patterns of viral replication both within groups (rabbits) and between groups. The basic problem with such scenarios being viewed as potentially decisive resolutions to the debate, however, is that it is fairly easy to construct explanations from either of these perspectives for whatever data is found or even merely envisaged. Lest this sound a little too *a priori* as a pronouncement of what we must find, let us consider a few good candidates for just the sort of decisive result we seek. Since the primary interest in the case is

whether it constitutes a decisive test for group selection, I focus on results that might be taken to offer such decisive support.

For example, consider the basic finding that virulence increases within hosts but decreases over the whole population over time. Surely, one might think, this at least prima facie supports the group selectionist view, since that view posits opposed forces of selection at different levels that directly explain the within- and between-group trends. Yet an individualist should respond that while individual selection should indeed increase virulence, faced with the environmental circumstances of shorter-lived hosts, it will also drive lower levels of virulence. That is, rather than seeing individual and group selection as competing forces that pull in different directions, as does the proponent of group selection, the individualist views both forces as being at the individual level. Each reflects a different adaptive pressure faced by individuals, with the one driving lower virulence winning out.

Likewise, consider experimental or naturalistic conditions that putatively reduce the effects of group selection and that thus lead to increases in levels of virulence. Lewontin had predicted that the introduction of the European rabbit flea, *S. cuniculi*, would increase virulence, presumably because doing so would effectively reduce the "groupishness" of viruses located on particular rabbits, and so limit the effects of group selection in promoting reduced levels of virulence by increasing its transmissability between rabbits. Prima facie, such manipulations would support the group selectionist explanation, in much the way that the findings of Michael Wade's celebrated flour beetle experiments that used an artificial group selection paradigm support group selection. In both cases, we manipulate factors that would adjust the strength of the force of group selection, were it to exist, and observe a phenotypic change that confirms the hypothesis that group selection is acting in the natural environment.[21]

There are two problems with this idea, one pertaining to the inconclusiveness of the empirical data relevant to Lewontin's particular suggestion, the other more general.

The first is that the effects on the virulence of the myxoma virus of introducing an alternate vector remain unclear. In part this is due to regional variation and the environmental sensitivities of this new vector. Studies at government sites around Canberra in 1977 and at Lake Urana in New South Wales in 1981 suggested that the introduction of rabbit fleas had little effect on the transmission of the myxoma virus, while results from South Australia in 1983 supported the effectiveness of the new vector in transmitting the virus. Temperature and moisture are crucial

variables here, and the mxyoma virus continued to be transmitted by the preexisting vector, the mosquito, in all three locations over the summer months.[22]

The second and deeper problem is that even had the empirical results been more clear-cut, they could also have been explained from the individualist's point of view. This is because the relevant manipulation changes the nature of the population-structured environment in which individual organisms exist, and with it the relationship between individuals and this environment. The explanation encapsulated in (1)–(3) would no longer apply, true enough. But that is because in the new environmental circumstances killing one's host is no longer detrimental to the fitness of the individual virus. So low virulence loses the fitness advantage it had in the pre-flea environment. In the new environment, increased virulence has a higher level of fitness, and so we would expect it to be the trait that evolved.

Consider a third candidate finding that might be thought to provide the decisive evidence sought. It is possible for a microorganism to have a high reproductive rate without killing its host. *Escherichia coli* bacteria, for example, are extremely rapid reproducers in the human gut, but do not usually pose a threat to the life of the host organism. In the case of the myxoma virus, there could be a strain of the virus that approximated *E. coli* in this respect, somewhat like how myxoma actually operates in *Sylvilagus* hosts. For example, a viral strain that increased the number of lesions on a rabbit, thus increasing transmissibility, with reduced infection of vital organs in the host (for example, due to host adaptation), would have this effect. Likewise, a strain that concentrated lesions on the head of the rabbit (where *S. cuniculi* concentrates, especially the ears) would have a similar transmission advantage. With an increase in transmissability, we could expect that, *ceteris paribus*, levels of virulence would increase.

I should like to make two points about the gap between this prediction and any conclusion about the level or levels at which such a selective force acts. First, the intricacy of the relationships between host, vector, and virus, and how each of these are affected by environmental parameters that are subject to change, makes it unlikely that all other things *will* be equal. Second, plausible explanations could be provided for such an outcome from each of these perspectives. Whether such an increase in virulence brought about by increased transmissability does so by virtue of selection operating directly on real population structures, groups of viruses, or does so by virtue of its operation on an individual's sensitivity to changes in its environment, would remain open.[23]

These examples indicate the sorts of problem with the appeal to decisiveness. There is, however, a principled reason why the problem here is general. We have two paradigms of evolutionary explanation, that of group selection and that of individual selection. Each has a basic repertoire of tools that can be adapted to much the same data sets in the myxoma case. Both can acknowledge the reality of a group structure (or a variety of group structures) within the overall virus population, and each accommodates this fact in its own way. The group selectionist sees natural selection as operating on the groups themselves, eliminating those groups that are less fit, which are groups whose members have high levels of virulence. The individual selectionist, by contrast, sees natural selection as operating on individuals sensitive to features of their environment, including the group structures imposed by the nature of the hosts and vectors present for transmission. If the resources of each view are rich enough to explain any putatively decisive result, as I have been suggesting, then this provides support for my second conclusion, that the myxoma case is in fact irresolvable vis-à-vis the question of how selection is really operating in it.

An aside for aficionados: it will not have escaped the notice of some that the reasoning here bears a similarity to that in Kim Sterelny and Philip Kitcher's response to the argument of Elliott Sober and Richard Lewontin that claimed that heterozygote superiority in the case of sickle cell anemia could not be explained by genic selection. Sober and Lewontin argued that, in this case, the smallest unit on which selection operates is the diploid genotype at a locus. Sterelny and Kitcher replied that individual alleles could be seen as agents of selection provided that one recognized the other allele in the genotype locus as part of the environment of the allele that is selected. If the parallel between this case and that of myxoma holds up, and what I have been arguing about irresolvability in that latter case is correct, then this debate over sickle cell anemia should also be irresolvable. End of aside.[24]

If the myxoma case is irresolvable, then it might seem a short step down the path to generalizations of this conclusion, perhaps even to the debate over group selection itself. But as I implied in the introduction, even this "short step" involves tasks that lie beyond the current chapter. For example, it would require more than the passing mention I have just made of the sickle cell case, as well as a discussion of other putative test cases of group selection, such as that of female-biased sex ratios. As a way of reinforcing my conclusion about the irresolvability of the myxoma case short of engaging in such discussions, and to move to some broader issues

that this conclusion itself raises, I focus next on a metaphor occasionally made explicit in the myxoma debate and otherwise often not far beneath the surface of the debate. This is the metaphor of particular agents of selection as having *standpoints* or *viewpoints.*

7. DECISIVENESS, VIEWPOINTS, AND AGENCY

Much of the myxoma debate can be cast in terms of talk of the viewpoints of particular organisms, and indeed has been. The conception of evolution in terms of the selfish gene has perhaps made most effective use of such viewpoint talk in evolutionary biology more generally.[25]

The primary viewpoint adopted in the debate over myxomatosis is that of the virus. Group selectionists hold that the viewpoint of the group of viruses that live on a rabbit is necessary for understanding, or sheds light on, just how natural selection operates in the myxoma case. We could encapsulate the concern that individualists have about the group selectionist account in terms of whether the viewpoint of virus groups adds anything to our understanding of the phenomena to that provided by the viewpoint of individual viruses. I propose to use this metaphor in conjunction with the details sketched in the previous two sections to raise a series of further complications.

The first is that the viewpoints of viruses (individuals or groups) are not independent of those of rabbits, especially given that the central concept in play, virulence, is often characterized relationally in terms of effects of the virus on the well being of rabbits. The second is that even these four viewpoints are not exhaustive, for there is that also of the vectors, as well as that of a nested hierarchy of groups, especially of viruses. As I implied in section 5, if rabbits constitute a group of viruses, then surely (given contagious infection) a hutch of rabbits does as well, as does the local deme of rabbit hutches. And given the differential transmissability of viruses located on different parts of the rabbit, all those viruses on the rabbit's head, or its ear, or around its eyes, also constitute groups of viruses. Note that here a variation on the arbitrariness problem from section 4 arises, for although rabbit-bound viruses are not an evolutionary arbitrary group, they are far from a unique such group. Thus, the focus just on rabbit-bound viruses as the object of group selection seems either arbitrary or in need of further justification.

On the group selectionist view, within each of these groups individual selection should promote increased virulence, while group selection should promote decreased virulence. Individual selectionists, of course, have their own account of how to understand this added complexity. The

third complication, in light of the first two, is that there seems to be no fact of the matter as to which viewpoint is (or viewpoints are) *the* correct viewpoint(s) for thinking about myxomatosis.

Consider now the viewpoint not of the virus but of the rabbit. When researchers found that rabbits had increased their resistance to the myxoma virus in the years following its introduction, this result was readily intelligible within the parameters of the theory of individual selection. What would have been paradoxical, in much the way as was the discovery of lower levels of virulence, would have been the finding that rabbits had decreased their levels of resistance. Although this was not found, I want to explore how this possible finding would be explained from each side of the debate over the myxoma case.

One initial thought is that if high resistance evolves by individual selection, then low resistance should evolve by group selection. (After all, this would parallel what has been said when considering the viewpoint of the virus.) To make the explanation of this explicit, consider the following argument sketch:

A. High resistance has a higher level of fitness for an individual rabbit.
B. But highly resistant rabbits spread the disease throughout the population.

Thus,

C. Groups of low resistance rabbits are fitter than groups of high resistance rabbits.

So,

D. Low resistance evolves by group selection.

Note that (A)–(D) provide an account of the evolution of low resistance in rabbits that closely parallels the group selectionist account of the evolution of low levels of virulence in the virus. In both cases, a trait that reduces within-group fitness nevertheless evolves because of group selection.

Low resistance would not, however, provide decisive support for the theory of group selection, since there is another explanation available that appeals only to individual selection. Again, to parallel the explanation of the evolution of low levels of virulence encapsulated in (1)–(3), consider the following individual-level explanation:

1.* A rabbit killed by a viral strain is less fit than one not so killed.
2.* Low resistance reduces the chances of being killed by a viral strain.

Thus,

3.* Low resistance is an adaptation of individual rabbits.

So,

4.* Low resistance evolves by individual selection.

Like the explanation of the evolution of avirulence by an appeal to individual selection, this explanation need not commit the averaging fallacy. Unlike that explanation, at least one of its premises seems strangely counterintuitive and stands in need of special justification.

This is (2*): how could low resistance increase one's fitness by reducing one's chance of parasite-induced death? Here an appeal to the sensitivity of individual rabbits to the nature of their environment needs to be made explicit. Given that the virus itself varies in its virulence, if the contraction of less virulent strains were to provide some immunization against the contraction of more virulent strains, then given other environmental conditions – such as the prevalence of less virulent strains – lower resistance could promote longevity in rabbits that have it (cf. also the sire effect, mentioned in section 5). This is how other pox viruses operate on humans, and why having chickenpox as a child is not altogether a bad thing. Fenner and Ratcliffe in fact present some evidence for just this sort of effect in myxomatosis, though they remain neutral as to whether this is caused by an interference effect between competing strains or a heightened immune response from the host.[26]

Alternatively, (2*) might be true, not by virtue of the structure of the viral environment and its interaction with individual rabbits, but because of facts about vectors and how they interact with rabbits with various levels of resistance. Infected rabbits may have less appeal for arthropod vectors than do uninfected rabbits, and so lower resistance may be a strategy that individual rabbits adopt in order to limit their exposure to highly virulent strains of the virus.

The counterfactual nature of the "finding" of the evolution of low resistance in rabbits should make it clear that this is intended as part of a "how possibly" rather than a "how actually" story. The interest of such a Just So Story is twofold in the present context.

First, it reminds us that we need to consider the full environment of an individual organism, including the nature of both its parasites and whatever group structure they have, as well as that of the vectors that mediate between host and parasite. In so doing, it draws attention to the toolkit of an individualist who appeals to the sensitivity of an individual to its (complicated) environmental circumstances. This reinforces the conclusion that such an individualist explanation will likely be available, once the empirical details of any such case are filled in, and so adds to the conclusions for which I have already argued.

Second, this "just so" story leads us fairly naturally into another – one that reminds us that there remains a further viewpoint, that of the gene. For we can now consider the following explanation of the "evolution of low resistance":

1.+ Genes whose vehicles are killed by viral strains in E are less fit than those in vehicles not so killed.
2.+ Genes for low resistance in E reduce the chances of one's vehicle being killed by viral strains.

Thus,

3.+ Low resistance is a genetic adaptation.

So,

4.+ Low resistance evolves by genic selection.

(1^+) – (4^+) make explicit the thus-far implicit appeal to an environment. I suggest that the genic selectionist defending (2^+) has at least as rich a repertoire of tools as has the individualist defending (2*).

If this is correct, then the basic problem for resolving the debate over the myxoma case is compounded, my conclusion about its likely irresolvability is further reinforced, and we have some additional reason to think that the extension of this conclusion beyond the myxoma case more generally is defensible. But this also raises two broader issues about the nature of selection and how we conceptualize it. To raise these issues as provocative questions: doesn't the irresolvability of the debate over the myxoma virus, and perhaps the levels of selection more generally, mean that the viewpoint of the individual and that of the group are simply two equivalent ways of viewing natural selection? And does the irresolvability claim here call into question the very conception of selection as operating at distinct levels? I shall say something briefly about these issues in closing this chapter.

8. How Deep Does Irresolvability Reach?

Near the beginning of this chapter, I noted that pluralism has some vogue as a position about the levels of selection. A prominent form of pluralism, one that has proven especially popular amongst a range of biologists and philosophers of biology, holds that there is an important sense in

which different models of selection are equivalent, such that the choice between them is to be made on pragmatic grounds. I shall call this view *model pluralism*, since it adopts a pluralistic stance toward various models of selection, including individual and group selection models.

Model pluralism provides a seemingly natural view to adopt if we accept either the specific irresolvability claim I have argued for about the myxoma case, or the more general irresolvability claim that I have groped toward. But despite appearances, the position I have defended in this chapter has little affinity with model pluralism. In fact, I find model pluralism prima facie implausible insofar as it implies that the disagreement between proponents of individual and group selectionist accounts of the myxoma case is merely apparent, heuristic, or pragmatic. The intuition that there is a fact of the matter about which these two sides disagree, one concerning the underlying biological ontology, runs deep. It should be given up only as a last resort.

What model pluralists are right about, and what is reinforced by our brief probe into the virological literature on myxomatosis in section 5, is that there is a deep interdependence between the various levels of selection. This presents a prima facie challenge to the predominant conception of selection as acting on distinct levels. Biological reality is perhaps more aptly conceived of as fused, enmeshed, or *entwined*, rather than hierarchically structured into neat levels, in that the properties on which selection acts, and indeed the mechanism of selection itself, do not come prepackaged at distinct levels. Conceiving of natural selection as operating at various "levels" would then be a simplification of the entwined, messy reality, a conception that imposes structure on, rather than simply reflects, biological reality. Reduced virulence in the myxoma virus can be viewed either as an adaptation of individual viruses or as a product of intergroup competition. This is not because model pluralism is true, however, but because the "levels" themselves are entwined.

The evolution of the myxoma virus in natural populations of rabbits in Australia is less conclusive as an example of group selection than proponents of group selection think. But the analysis of the example provided by their individualistic critics does not win the day either. Rather, the debate here stands as an example of a scientific dispute that cannot be resolved by the current evidence, and I have argued that it seems unlikely to yield to further evidence. Reflection on some of the complexities to the phenomenon of myxomatosis reinforce rather than ameliorate the irresolvability of the myxoma case. Thus, there are reasons to think that the scientific dispute here cannot be rationally resolved in a conclusive manner.

This conclusion almost certainly generalizes to other cases invoked in the debate over the levels of selection. While one might think that it generalizes to the whole debate between proponents of individual and group selection, that would require significantly more and I think a somewhat different argument than I have provided here. Those who endorse such a view of the general debate over the levels of selection may be tempted by model pluralism, but this is not my temptation. Rather, I think that the argument here raises questions about the adequacy of the very conception of natural selection operating at distinct levels, a conception ubiquitous in debates over how natural selection operates. If this is correct, then perhaps we need to rethink some large issues in the field.

Some of those issues concern pluralism, the notion of entwinement, and their relevance to the agents of selection. In the final chapter, I turn to these issues and the claims that I have made about them in this section.